TI Identification of the transferrin receptor as a novel immunoglobulin (Ig)A1 receptor and its enhanced expression on mesangial cells in IgA nephropathy.
AU Moura I C; Centelles M N; Arcos-Fajardo M; Malheiros D M; Collawn J F; Cooper M D; Monteiro R C
SO The Journal of experimental medicine, (2001 Aug 20) Vol. 194, No. 4, pp. 417-25.
Journal code: 2985109R. ISSN: 0022-1007.
AB The biological functions of immunoglobulin (Ig)A antibodies depend primarily on their interaction with cell surface receptors. Four IgA receptors are presently characterized. The FcalphaRI (CD89) expressed by myeloid cells selectively binds IgA1 and IgA2 antibodies, whereas the poly-IgR, Fcalpha/muR, and asialoglycoprotein receptors bind other ligands in addition to IgA. IgA binding by mesangial cells, epithelial cells, and proliferating lymphocytes is also well documented, but the nature of the IgA receptors on these cells remains elusive. A monoclonal antibody (A24) is described here that specifically blocks IgA binding to epithelial and B lymphocyte cell lines. Both the A24 antibody and IgA1 myelomas bind a cell surface protein that is identified as the transferrin receptor (CD71). The transferrin receptor selectively binds IgA1 antibodies, monomeric better than polymeric forms, and the IgA1 binding is inhibitable by transferrin. Transferrin receptor expression is upregulated on cultured mesangial cells as well as on glomerular mesangial cells in patients with IgA nephropathy. The characterization of transferrin receptor as a novel IgA1 receptor on renal mesangial cells suggests its potential involvement in the pathogenesis of IgA nephropathy.

L5 ANSWER 24 OF 25 MEDLINE on STN
TI The role of non-immune IgG in controlling IgG-mediated effector functions.
AU Segal D M; Dower S K; Titus J A
SO Molecular immunology, (1983 Nov) Vol. 20, No. 11, pp. 1177-89. Ref: 88
Journal code: 7905289. ISSN: 0161-5890.
AB The majority of evidence supports the conclusion that IgG-dependent effectors respond to antibodies which have been polymerized artificially or by polyvalent antigens, but not to monomeric IgG antibodies. Effectors can distinguish polymerized IgG antibodies from monomeric IgG because they contain multiple receptor units and can interact multivalently with polymerized IgG. However, monomeric IgG is present at very high concns in plasma and interstitial fluids and will inhibit multivalent interactions in vivo between polymerized antibody and effectors. Such inhibition raises the question of how IgG-mediated effector responses could function in vivo. In this review we present a mathematical model which quantitatively predicts how polyvalent ligands interact multivalently with receptors in the presence of excess monovalent ligand. We then show that results from experiments in vitro using such diverse systems as the binding and endocytosis of immune complexes by macrophages, complement-mediated lysis of antibody-coated target cells, and ADCC can be explained qualitatively by the model. We conclude that monomeric IgG does not totally inhibit IgG-mediated effector functions but, rather, raises the threshold of antibody binding which is required to elicit a response. We then consider how non-immune IgG may serve as a homeostatic regulator of IgG-dependent responses, in vivo, perhaps for the purpose of inhibiting responses to low levels of cell-bound IgG autoantibodies.

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     FILE 'MEDLINE' ENTERED AT 10:14:25 ON 25 APR 2008
L1            0 S POYMERIC (8A)ANTIBOD?
L2          187 S POLYMERIC (8A)ANTIBOD?
L3           37 S L2 AND (APOPTOSIS OR RECEPTOR)
L4         1597 S POLYMER?(5A)ANTIBOD?
L5           25 S L4 (8A)(RECEPTOR OR APOPTOSIS)
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